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I.

GENERAL ASPECTS OF THE BRAIN ANATOMY OF THE FEEBLE-MINDED.

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ABSTRACT.—Small amount of intensive anatomical work available in the literature (Bourneville, Hammarberg, Tredgold, Bolton). Research emphasis on other aspects (heredity, mental tests, social service, education).

Situation in the biology of feeble-mindedness (Ballantyne). The question whether any feeble-minded persons have normal-looking brains. Relative absence of work on the brains of feeble-minded of higher grades. Modern knowledge of cortex topography.

Tredgold's distinction of primary and secondary amentia ("germinal blight" versus "arrest"). Classification of W. W. Ireland. Exogenous nature of numerous cases (intrauterine lesions). Tredgold's view that germinal variations are pathological and not spontaneous. The suggestion that germ cells may be poisoned. Brain findings in exogenous cases.

Anatomical classification, Bourneville, 1889; Bourneville's researches. Hammarberg's report on 9 cases (psychic defects connected with defective nerve cells in the cerebral cortex). Idiocy thought to be due to developmental check in later foetal life. Marked weak-mindedness thought to be due to arrest of development in a stage normal for the first years of life. A third group with focal factors of inhibition occurring during the first year of life.

Most of Hammarberg's work devoted to the microscopy of the *normal* cortex. Necessity of recognizing certain shrinkage processes as artificial. Hammarberg's claim that disorderly arrangement of cortical cells is also found in normal material (undeveloped cells frequently taken for atrophic ones).

Summary of Hammarberg's work with 4 cases of idiocy, 2 of marked weak-mindedness, 3 of slighter degrees of weak-mindedness.

Tredgold's conclusion that the essential basis of primary amentia is cellular, due to an imperfect or arrested development of cerebral neurones. Tredgold's belief that irregular arrangement of cells is characteristic of feeble-mindedness material.

Situation in the anatomy of feeble-mindedness as presented at the Medical Congress in Paris in 1900.

Work of Mierzejewski (anatomical lesions are developmental deviations). Mierzejewski's special group of cases in which the gray matter is disproportionately large as compared with the white matter. Heterotopia.

Classification of Shuttleworth and Fletcher-Beach; microcephaly probably not caused by premature synostosis of cranial sutures. Congenital hydrocephalus extends the lateral ventricles lengthwise, acquired vertically and transversely (Meynert); scaphocephaly; Mongolian idiocy; other groups.

Syphilis not a common cause of feeble-mindedness; juvenile general paresis as first observed by Clouston; traumatic cases; so-called hypertrophy.

American literature. Wilmarth's study. 25 cases in 100 yielded no brain lesions to gross analysis. Quotations from Wilmarth's conclusions.

Problems suggested by the literature of the brain anatomy of the feeble-minded.

We present herewith the first section of the Waverley Researches in the Anatomy of the Feeble-Minded begun in 1913 under the auspices of the Massachusetts School for the Feeble-Minded. This first section of Researches contains

I, a general introduction to the topic of brain anatomy of feeble-mindedness,

II, a particular description of ten instances of feeble-mindedness of various grades, together with autopsy and microscopic findings, and

III, attempts at correlating the brain anatomy and histology with clinical and especially with psychometric (mental test) findings. Further studies are under contemplation with the object of gradually enlarging the brain series, from which, when it shall have contained say 50 brains, we may be able to draw sound conclusions.

Let us note at the outset our surprise at the extraordinary neglect into which this topic has fallen. Bourneville, Hammarberg, Wilmarth, are the older names of workers contributing carefully sifted cases studied in the interest of feeble-mindedness. Most authors deal with feeble-mindedness as a stalking-horse in some other scientific pursuit, teratological, embryological, anatomical, histological, etc. We claim for the present work that, despite numerous theoretical and ancillary interests, *we have studied feeble-mindedness from the standpoint of feeble-mindedness*. In particular we have tried to place our cases in a tentative order of mental strength, correlating brain appearances with mental tests. Our purpose has been at least as much to evaluate the mental tests by throwing their results against the brain pictures as it might have been to evaluate the brain pictures in the light of mental tests. We may anticipate our conclusions so far as to say that, roughly speaking, the architectonic order in which the brains arranged themselves corresponds to the psychometric order indicated by the mental tests.

Thus, we must emphasize, we are not here studying the brain for the brain's sake, or the mental tests for their sake alone. We have no eugenic, economic or social interests paramount in the present inquiry.

This first series of ten cases is intended as an orienting and introductory series to a more extended study which at present contemplates the examination by modern methods of 40 more cases, to make up a series of 50, from which series it may be safe to draw provisional conclusions. The first series has accordingly been made to include all available material from several sources which was found to comport with certain necessities of modern research in feeble-mindedness.

One might readily get the impression that the literature would be found to contain enough properly examined material from which to draw conclusions. One would have in mind the Bicêtre reports of Bourneville, beginning in 1881 and continued at successive intervals for 20 years. One would have in mind the masterly work done in Sweden by

Hammarberg and published after his death at Upsala in 1895. Another source of information would be the work done by Dr. A. F. Tredgold under the London County Council early in this century and published in Mott's Archives of Neurology in 1903, embodied in Tredgold's book on Mental Deficiency, second edition, 1914.

Then one would find the work of J. S. Bolton "On the Histological Basis of Amentia and Dementia," published in the same volume of Archives of Neurology and later embodied in a more popularized form in Bolton's book "On the Brain in Health and Disease," London, 1914.

In the textbooks on feeble-mindedness, one would find numerous references to Italian work in anatomy. In various textbooks one would find references to considerable work on the anatomy of feeble-mindedness (see for example, the literary references in Ziehen's account of "Diseases of the Brain and Meninges in Childhood," 1912).

In point of fact, however, the anatomy of feeble-mindedness has not been attacked systematically over a period of years having due regard not only to clinical features, but to anatomical and histological features.

On the whole, the remarkable developments in education of the feeble-minded following the initial work of Séguin have developed almost independently of the anatomical, histological and physiological sides of the topic. With the developments of recent years a tremendous emphasis has begun to be laid upon the hereditary and eugenic aspects of feeble-mindedness, and a large effort has been expended upon certain applications of psychology to the mental testing of the feeble-minded. But neither the hereditarians nor the applied psychologists have particularly concerned themselves with the broader biological aspects of the whole topic. We must confess that on the whole, the broader biological aspects of feeble-mindedness have tended to be concealed in the mass of practical applications which the mental testing methods have unfolded and the web of promises spun by the eugenicist. Accordingly we face a situation with the feeble-minded in which their education, their heredity, and the methods of their mental testing have absorbed interest. In the spring of 1915, however, the note of research in this field was struck at the National Conference of Charities and Correction, perhaps somewhat to the astonishment of the listeners, who have been far more interested in social service than in the more recondite aspects of research. However, educational research, eugenic research, research in applied psychology, and sociological research, all need effort on the biological basis. It seems as if this basis had been sadly and almost inconceivably neglected in the past.

What is the present situation, biologically speaking? The *biologist*, if asked the nature and measure of his interest in feeble-mindedness, would be apt to answer some-

what as follows: The institutions might supply us with a vast deal of material of the greatest interest from such a standpoint, for example, as that of Ballantyne, whose fundamental collection of material on "Antenatal Pathology and Hygiene" was published in 1902 and 1904 respectively. There should be much, the biologist might continue, of great interest from the standpoint of monstrosities, and the teratologist might be able to unravel any number of intricate problems of the mechanics of development from the anatomical material of institutions for the feeble-minded. He would have to consider not merely the immediate pathology of the antenatal period, but also the postponed effects which Ballantyne has emphasized, and not only the postponed effects in the neonatal period of life, but also its postponed effects upon still later life.

Ballantyne was able to determine important relations of antenatal pathology to no less than seventeen departments of science (see his figure 3, page 18). Of the considerations in Taruffi's *Storia della teratologia* would come an application, and the further development in studies of the idiot types with the methods available to modern research. For example, the microscopy of all types of terata, described in chapters 14-30 of book 3 of Ballantyne's volume on the Embryo would get a new importance if proper histological studies were carried out. But withal, the teratologist would be studying this material from the standpoint of teratology and tissue mechanisms, and the fate of the feeble-minded as such would not be visibly altered for the better in a long period of years.

The *pathologist* might see not only the importance of antenatal pathology in the direction of monstrosities, but he might be greatly interested in what Ballantyne has termed the intrusion of the antenatal factor in neonatal disease as shown by instances of intracranial traumatism and intranatal infections (ophthalmia neonatorum, etc.). The pathologist would also be interested in the neonatal infections, such as sepsis neonatorum, as also very particularly in what Ballantyne has called the disturbed antenatal readjustments, such as icterus neonatorum, and the like. Here again the interest in the anatomy of the feeble-minded would be an interest derived *ab extra*. The pathologist must go to the specialist in feeble-mindedness for his material and carry his interest to that material. Hence from the pathological side it can hardly be hoped that there will be a tremendous flow of new work on this topic.

Again, the *embryologist* must be interested in the subject, as is clear throughout both volumes of Ballantyne's work; but the embryologist has so far shown little interest in what has sometimes been termed "late" embryology, confining his attention to the plentiful and unsolved problems of the earlier months. Hence what happens to the hypoplastic organ in the *viable* victim of feeble-mindedness is of less immediate interest to the embryologist by reason of the embryologist's comparative ignorance of late embryology

in general and hence of the appearances in organs sufficiently developed to permit their bearers to live.

It might be thought that the *physiologist* would take a tremendous interest in a number of aspects of feeble-mindedness since he would perhaps be able to find the differential effects of imperfect glands of internal secretion and a variety of simplified phenomena of nervous and circulatory reactions. On the whole, however, the physiologist probably feels that his knowledge of these matters in laboratory animals remains too tenuous to permit carrying the standard far afield in feeble-mindedness.

The *zoölogist* and even the botanist of course have the greatest interest in the germ cells, their development and interferences with their development. In various writers one discovers tendencies to the thought that there is a pathology of the germ plasm which at first sight spoils the simplicity of the Weismannian doctrines about the immortality and comparative immutability of the germ plasm. The interest in eugenics has fortified this interest, and no doubt all the germ cell biologists as well as the eugenists have fully in mind the value of studies in the ancestry and familial traits of the feeble-minded. Nevertheless these interests have not crystallized into a theory or plan of attack as to feeble-mindedness.

Like the child in Kipling, each science or art above mentioned has mysterious errands of its own; hence it seems likely that the workers in feeble-mindedness themselves must organize their own researches. Where then should research in feeble-mindedness begin? There is no lack of interest in the education of the feeble-minded. There is no lack of interest in various forms of social service, whether it takes shape as help for the individual, aid to the public purse, or an attempt to safeguard society. There is no lack of interest in heredity and eugenics. There is no lack of interest in the general administrative problem which offers one of the most productive regions for the administrator in the whole field of public service. There is no lack of interest in applied psychology as it has culminated in such mental tests as those of Binet, de Sanctis, Healy, Terman, and Yerkes. Little, however, has ever been said in favor of a consistent attack on the anatomy and physiology of the feeble-minded.

The present researches form a comparatively modest introduction to that highly desirable attack upon the physical aspects of feeble-mindedness which have been so inconceivably neglected in the past. We feel that it is wise to concentrate attention upon brain anatomy for the present, despite the fact that the ultimate value of physiological work, notably in the field of the glands of internal secretion may be immeasurably greater. However, in a number of fields of neuropathological and psychiatric research, we have found that much time can be saved if the physiologist and clinician can have the advantage

of knowing whether they deal on the one hand with entirely abnormal brains or on the other hand with relatively normal ones. This dichotomy of the material is rather apt to leave one with three groups of material, — a) material with gross lesions of an obvious and striking nature; b) material with lesions of a doubtful and questionable character, and c) material without obvious evidence of disease. The worker, whether he deal with insanity, epilepsy, criminal anthropology or any other major field touching the nervous system, finds it convenient to utilize various fundamental studies in the two limiting groups in the series, viz., the group of cases with obvious gross lesions and the group of material with no obvious lesions. He must leave the intermediate or non-interpretable group on one side at the beginning of his research.

The topic of feeble-mindedness is rendered difficult by the fact that the observer apparently cannot compare abnormal material with normal looking material just by reason of the absence of any normal looking material. At least an observer like Tredgold remarks that he has never “yet seen the brain of an idiot, a low or even medium grade imbecile which could be regarded as normal upon careful naked-eye examination” (of course it will be noted that Tredgold does not here speak of the imbeciles of higher grade and of the feeble-minded proper). It is safe to say that from the literature one cannot for the present assert whether or not the brains of imbeciles of higher grade, of the feeble-minded proper or so-called morons, or of the dullards or simple-minded persons who grade somewhat higher than the former, have normal looking brains. Beyond question, however, most workers distinguish at least roughly between the idiots and imbeciles of lower grade who are virtually bound to show obvious brain lesions, and the higher grades of feeble-minded, who may or may not show such lesions. In either case there is a tendency to the belief that amongst the higher grades will be found persons whose feeble-mindedness tends toward functionality as a limit. There is a tendency to the belief that the higher grades of feeble-minded are persons whose brains are fairly well developed in the sense of having a full complement of cells like their normal brethren, but whose cells do not for some reason work properly or have not, for some reason, developed their full panoply of processes. That there is a group of feeble-minded subjects that in a sense correspond to idiopathic epileptics or to the so-called functional psychopaths (for example, victims of manic depressive psychosis of modern conception), or like the delinquents whose brains are working, as it were, perversely, without structural basis for the perversity, amounts even to a working hypothesis with many observers. Upon this working hypothesis is founded a number of conceptions as to the educability and comparatively good prognosis of certain feeble-minded subjects “under proper conditions” or with an “improved environment.” Whether there is an environmental group

of feeble-minded subjects, whether there is, as it were, an idiopathic or functional group of these cases, is an open question. Just as we suspect that there are frank psychoses which have their bases not in structural brain disorder but in exogenous factors working from outside the brain, so we may theoretically conceive that there are feeble-minded subjects in the same logical group. However, in a general way, the concept of "discords played on a good instrument" is not a concept which appears to have a wide scope in feeble-mindedness.

The literature does not lack work of a systematic and comparative nature upon idiocy. One has only to remember the work of Hammarberg above noted. The literature lacks consistent studies of feeble-mindedness of higher grade from the anatomical and histological points of view. This task should be one of the major ones of the present undertaking. Hammarberg's work was one of the first pieces of work not only in the anatomy and histology of feeble-mindedness but in the topographical histology of the nervous system. The topographical histology of the nervous system has only been thoroughly staked out within the present century. The idea of the brain's differential topography doubtless lay completely in the minds of such workers as Bevan Lewis, Betz, Meynert, Cajal and Nissl in the nineteenth century. Early in the present century, systematic works like those of Campbell, Brodmann and Cajal began to appear and to have currency. These works on topographical histology were supported by such brain anatomists as Elliot Smith and Ariens Kappers, as well as by the comparative anatomist, Edinger. The idea of cerebral cortex mapping, of a kind of cartography of the brain, has now obtained a firm foothold in neurology and neuropathology. It must, however, be accredited to the science of feeble-mindedness that the lamented Hammarberg was one of the first histologists who saw the importance of such cortex mapping and began his work upon the material of idiocy. To be sure it will be many years before a properly equipped institute carries out the inchoate aims of Hammarberg and the aims which we can all now so clearly see when we associate in conception the material of feeble-mindedness with the modern differential work on cortex structure.

One of the most fruitful distinctions in feeble-mindedness of the present day is that of Tredgold, who, seeing the inadequacy of the distinction of the feeble-minded into congenital and acquired cases has replaced this dichotomy with a new and different one, namely, with a division into what he calls primary and secondary amentia.

The primary aments are victims of what is sometimes termed "germinal blight," whereas the secondary aments are victims of some cause working from outside the brain. The primary ament, according to Tredgold, has cells with an intrinsic vital deficiency,—cells which are unable to develop. The secondary ament is the victim of arrest, due to

some external cause. One might say that the cells of the ament, if this distinction of Tredgold can be upheld, have "run down" or ceased to develop at a certain point, whereas the cells of the secondary ament have not run down of themselves, but have run counter to some agent; that is, the cells have not ceased to develop, they have been stopped. The distinction reminds one of the simple nomenclature apparently introduced into neurology by Moebius, namely, the distinction between cases of endogenous and cases of exogenous origin. One deals, as it were, with a plastic impotence, an agenesis, in the case of the primary ament; one deals with a sort of plastic arrest or aplasia in the case of the secondary ament.

The value of this distinction of Tredgold's is that the older heterogeneous anatomical classifications of the feeble-minded, such as were, for example, available to W. W. Ireland in his book on the "Mental Affections of Children" can be supplanted by a simpler classification with a number of new research possibilities. Ireland, for instance, speaks of genetous idiocy, namely, idiocy in which the disease condition which entails deficiency of mind is complete before birth, and in which accordingly the presumption of the hereditary connection is stronger than in other forms. Another chapter deals with microcephalics, a third with hydrocephalics, a fourth with eclamptic idiots, a fifth and sixth with epileptic and paralytic idiots, and further chapters with traumatic, inflammatory, sclerotic, syphilitic and cretinistic idiots, and the last chapter with what Ireland terms idiocy by deprivation (namely, that condition of mind in which a child remains who is deprived of two or more of the principal senses).

According to Tredgold a great number of the intrauterine causes of feeble-mindedness are, as it were, equally exogenous to the feeble-mindedness as would be a cause operating early after birth. The antenatal and neonatal pathology of feeble-mindedness would have to be separated into a congenital and acquired group forthwith, unless we adopt Tredgold's distinction and consider that numerous antenatal causes, such as foetal sepsis, foetal syphilis, and the like are just as secondary in their effects as would be these conditions in the neonatal period. Tredgold accordingly distinguishes an intrinsic from an extrinsic group and would probably subscribe to Moebius' terms "endogenous" and "exogenous" as equivalents of his own. In his original discussion of amentia in Mott's Archives in 1903, he had already practically distinguished the intrinsic forms as due to the condition of the germ plasm and the extrinsic forms as due to the environment, which environment he divided into antenatal, intranatal and postnatal. But in his textbook Tredgold states that the real question is as to the respective influences exerted by heredity and by environment and proposes that the terms "congenital" and "acquired" be given up. Tredgold defines extrinsic causes as any factors of the environment which are cap-

able of prejudicially affecting the brain development of the offspring after conception has taken place, whether these factors be intra or extra-uterine. Amentia due to such causes, Tredgold terms extrinsic or secondary amentia.

Tredgold's employment of the term "hereditary" is possibly somewhat special to him. Primary amentia Tredgold regards as not "due to absence or suppression of some specific germ determinant" such as some Mendelians assert, but "as resulting from a diminished germinal vitality in consequence of which development tends to be incomplete." Inheritance takes the form of the "neuropathic diathesis or innate predisposition to neurotic imperfection." As to the origin of the germinal impairments that lie at the foundation of the intrinsic or primary group of Tredgold, we have a number of possibilities. The Royal Commission on Feeble-mindedness decided that "both on the ground of fact and of theory, there is the highest degree of probability that feeble-mindedness is usually *spontaneous* in origin, that is, not due to influences acting on the parent — and tends strongly to be inherited." Tredgold regards this as a confession of ignorance. He, however, looks upon the hypothesis of Davenport that "feeble-mindedness is not a new variation, but a perpetuation of strains defective from the beginning" as not tenable. Tredgold regards the germinal variations in the parents of the feeble-minded as pathological variations, as a vitiation and not spontaneous. He regards this vitiation as primarily due to the action of the environment, quoting the work of Paul on lead poisoning, the work of Féré on the effect of alcohol on incubating eggs, the more recent work of Stockard upon guinea pigs and other similar work. Not only may the germ cells be poisoned, but they may be altered by a variety of physical conditions, such as temperature and non-toxic chemical agents. Whether or not Tredgold's conception of the causes of what he has termed germinal blight are as he thinks environmental, there can be no doubt that it is extremely valuable to separate out all definitely exogenous cases from a residue whose cause may be left undefined.

What now can we see in the brains of primary as opposed to secondary aments? In the secondary types of feeble-mindedness, we shall often see effects of inflammation, appearances like that of porencephaly, hydrocephalus, and the like, which must appeal to us as flowing from causes operating from without and indicating no essential vital deficiency or vitiation of germ plasm, and we shall be greatly interested to determine the nature of these exogenous processes and the quality and extent of their effects upon the brain map. In the light of these irregularly deficient brains we should, under ideal conditions, be immensely interested to learn whether the mental states of the bearers of these brains also show functional cut-outs and losses to correspond with the irregular defects of the brain lesion. A tremendous number of neurological problems will be settled

and a deep vista of new problems can be predicted. Moreover, we should learn much by extended study of these secondary cases from the standpoint of what is termed in the German "Korrekturbildung." We shall namely be interested in discovering what effects may be wrought by the lack of structure A upon the condition of structure B which, under normal conditions, grows in structural and functional relation to A.

But suppose there be no demonstrable exogenous processes and no basis for supposing an exogenous process in a given case. What now will be found in the brain?

The literary sources of information from which to answer this question are not numerous. We must first resort to Bourneville's volumes on Researches in Epilepsy, Hysteria and Idiocy, published in the form of Reports from the Bicêtre, beginning in the year 1881. These reports contain numerous cases reported as fully as the state of science permitted. The majority of the autopsy reports in the earlier years deal with epileptics. Numerous remarkable cases of tuberous sclerosis (sometimes called Bourneville's Disease) are described and photographed. The majority of these cases of tuberous sclerosis were victims of epilepsy or of convulsions in some form. Numerous reports are scattered through the series dealing with myxedematous idiocy. Through the eighties Bourneville's reports contained few instances of case reports in which convulsions were not prominent. In the nineties began to appear more numerous reports of idiocy and epilepsy without convulsions. Special studies of hydrocephalus, porencephaly and microcephaly appear, and the term "symptomatic idiocy" is frequently used in connection with such cases.

In 1891 Bourneville presents in his 11th volume an anatomical classification of idiocy (communicated at the International Congress of Mental Medicine in 1889) which classification has 8 chief heads, as follows:—

Idiocy symptomatic of hydrocephalus; of microcephalus; of arrest of convolutional development; of congenital malformation of the brain (porencephaly, absence of corpus callosum, etc.); idiocy symptomatic of hypertrophic or tuberous sclerosis; of atrophic sclerosis (A, of one or both hemispheres, B, of one brain lobe, C, of isolated gyri, D, of *selérose chagriné du cerveau*); idiocy symptomatic of chronic meningitis or meningo-encephalitis; and finally, idiocy with pachydermic cachexia (myxedematous idiocy connected with absence of the thyroid gland). It can readily be seen how much of this classification, which is based upon fifteen years of work at the Salpêtrière and at the Bicêtre, has relation to those aments whom Tredgold would readily call secondary aments. Bourneville and his collaborators have published excellent photographs of many of the cases in this series of volumes. From time to time, histological descriptions of a relatively elaborate nature accompany the reports and deal with questions of neuroglia prolifera-

tions and the like in quite the modern manner, which is the more remarkable since much of the work was done before the vogue of neuroglia studies begun by the publication of Weigert's book on Neuroglia in 1895.

The reports of the nineties are filled especially with accounts of idiocy symptomatic of atrophic sclerosis and of chronic meningitis. Reports upon congenital idiocy are on the whole infrequent in Bourneville's volumes. Such a case published in volume 13, 1893, by Bourneville and Dauriac was, for example, a congenital epileptic idiot, which was almost completely asphyxiated at birth, began to walk at 8 and completed dentition at 10. This idiot had had convulsions at 8 months followed by incomplete paralysis of the right side. Contractures of all limbs with athetotic movements had supervened. Double craniectomy was performed for therapeutic purposes in the thirteenth year. The autopsy showed, besides the effects of the operation, a cerebral atrophy (weight 855 grams), or as Bourneville says, the autopsy findings may perhaps better be said to have confirmed the diagnosis "arrest of brain development" made during the patient's life. There were no areas of sclerosis or induration and this fact is taken by Bourneville to corroborate the clinical evidence as to the absence of any severe convulsions. There were, however, certain leathery appearances in some gyri, notably in the left parietal and frontal regions and in the right frontal and temporal sphenoidal regions.

From such a description and from Bourneville's own remark as to the possible diagnosis of cerebral atrophy, it can readily be seen that such a case very possibly belongs in the group of secondary aments of Tredgold, although the condition may well have been congenital in the usual acceptance of that term.

In 1895 Bourneville and LeNoir published an example of so-called complete congenital idiocy with paraplegia, contracture and deformity of feet. The brain of this case is described by Bourneville as having been reduced to the greatest simplicity, "virtually down to the elementary convolutions." Although these convolutions are in general voluminous, even strikingly voluminous, and the sulci are of a proper depth, annectant convolutions are almost completely lacking. Plates are presented of this case which died at 18.

In 1896 Bourneville and Ruel present another case of "complete idiocy," probably congenital. The brain showed neither meningitis nor focal lesions.

In 1897 Bourneville and LeNoir again published a case of congenital idiocy due to arrest of development, and state that the brain reveals no macroscopic lesion. Nevertheless its morphology is rudimentary and the arrangement of the convolutions is in many places abnormal. These might serve as examples of the few available reports concerning cases of so-called developmental arrest to be found in Bourneville's series.

Although he terms these cases congenital and speaks of some of them as victims of developmental arrest, they seem on the whole to stand nearest to the cases which Tredgold would be inclined to term primary aments. The number of cases of myxedematous idiocy and of various forms of so-called symptomatic idiocy with atrophic sclerosis and meningitis continue far to outnumber the cases of congenital nature. In passing one can only comment with admiration upon the breadth of Bourneville's general plan of attack on his problem, combining as it does the clinical, therapeutic and anatomical ideals.

We shall now naturally turn to Hammarberg for evidence as to the essential nature of these two groups of cases, whether on the one hand we adhere to the older distinctions of congenital and acquired cases, or adopt the more modern distinction of primary and secondary amentia proposed by Tredgold.

Hammarberg's monograph was published in Upsala in 1895, edited by Henschen, after the death of the author from appendicitis. His work appears to have been done a few years after his graduation in medicine and to represent a comparatively brief period of intensive endeavor. His report deals with 9 cases. He believed that he could prove in all cases that the psychic defect could be connected with defective nerve cells in the cortex. The cortical deficiency he considered to be due either to a standstill in development at some antenatal or early neonatal stage, in consequence of which but a small number of cells were permitted to arrive at high development. He admitted the possibility that cells might be destroyed during the process of mal-development.

In some cases development had been checked in small regions by agents working in a limited and local manner, but in such wise that the total development of the cortex had been unfavorably influenced. If the check in development occurred in the latter stage of foetal life, psychic development in the subject became impossible and these cases are grouped as idiots, "*blödsinnig*", in group A. Since the inhibiting factor acted unequally in different parts of the developing brain, a variety of disorders in the cranial nerves in locomotor capacity and in sensibility, such as were found in many cases, were explicable.

In a second group of cases the cortex had not been permitted to arrive at a degree of development more than one normal for the first years of life. These are cases which Hammarberg groups as markedly weak-minded (*in hohem grade Schwachsinnige*). Such cases also exhibited the same irregular cranial motor and sensory disorders.

A third group of cases is a group in which the inhibitory factor is thought to occur during the first year of life and to affect only a small region of the brain. The rest of the brain is regarded as developing properly in all directions, except that the number of cells seems to remain smaller than normal. The psychic result is that the patient is

inactive and less developed than a normal person of the same age. These are cases classified under B 2, as moderately and slightly weakminded (*mässig und in geringem grade Schwachsinnige*). Similar peripheral disorders are found in these cases also.

The idiots of Hammarberg's series number 4,

The markedly weakminded, 2,

The moderately and slightly weakminded, 3.

Although there are a number of drawings of gross brains in plates 6 and 7 of Hammarberg's monograph, the chief direction of Hammarberg's research is microscopic. Excellent drawings to scale of preparations from various areas of the cortex, stained by methylene blue, are presented in plates 1 to 5. Plates 1, 2 and 3 deal with the normal cortex (since it must be remembered that Hammarberg's work antedated the cerebral topography of late years) and deal with superior and inferior frontal gyri, with the precentral gyrus, the superior temporal, the superior and inferior parietal and superior occipital gyri, the central lobe, hippocampal gyrus, the fascia dentata, cornu ammonis, fascia cinerea, the gyrus cinguli and the gyrus centralis superior.

The pathological plates are 4 and 5 and contain 15 drawings to scale of a number of the above mentioned areas. The whole work is but a torso of what Hammarberg would doubtless have accomplished had he lived.

A number of Hammarberg's observations as to the microscopy of feeble-mindedness exhibit a very modern insight. For example, he dismisses the frequently expressed notion that the pericellular and perivascular spaces are enlarged about the brain cells of idiots, whether in consequence of lymphatic stasis or of cellular atrophy. He regards these features as shrinkage processes, finding them to occur only in material which had at some stage been placed in alcohol of marked concentration, and finding similar changes to occur in normal persons. These findings, then, are dismissed by Hammarberg as artefacts, and it is probable that all modern workers will agree with him.

A second statement to be found in the literature preceding Hammarberg and to some extent since his day is that the brain cells of the cortex in idiocy present a disorderly or imperfect arrangement, so that the apical processes lie at right angles to their normal direction or obliquely. Hammarberg discovered that identical appearances might be found in entirely normal material, noting especially that in the inferior frontal gyrus and the hippocampal gyrus such apparent disorder in the orientation of cells is normal.

A third question of contention in the literature deals with the occurrence of cellular atrophy, vacuole formation and pigmentary degeneration, but such changes as these were found by Hammarberg in but one of his 9 cases, and he notes that undeveloped cells have

frequently been taken for atrophic ones, especially if at the same time an imperfect technique has produced the artefact of dilated pericellular spaces.

Hammarberg accordingly dismisses the idea that the nerve cells of the idiot and imbecile do not fill enough space in the cerebral cortex in which they are embedded, that they are especially subject to irregular orientation therein, and that they are necessarily subject to atrophy, vacuole formation or pigmentary change.

Hammarberg's conclusions then deal with four cases of idiocy, namely, subjects deficient in all the higher psychic functions and incapable of psychic development. In three of these cases the greater part of the cortex was in a stage of development normally found in the last half of intra-uterine life. In a fourth case development had, according to Hammarberg, progressed farther and had reached a stage found normally in the first year of life; but in this case a process had occurred which had reduced development back to the same stage as in the other three cases.

The second group of cases, two in number, is a group of markedly weak-minded subjects, whose mental life corresponds to that of normal individuals of one to five years of age. Their cortex was found, according to Hammarberg, to be in a stage of development found in normal individuals during the same years.

The three cases of the group of moderately or slightly weak-minded are cases in which the mental functions are sluggish and somewhat less developed than in normal individuals of the same age. Such subjects are capable of a certain amount of education. Their degree of development, from a mental standpoint, might readily be compared to any special stage of development in normal individuals. According to Hammarberg, the number of nerve cells in the greater part of the cortex is much less than normal and the remarkable feature of the general brain situation is that in one particular region the cortex will be found to be in a stage of development normally appearing in the very first year of life. Here then a number of claims appear which would be well worth substantiating by extensive studies.

The most elaborate recent statement concerning feeble-mindedness is that by Tredgold in his book on "Mental Deficiency". The ideas are largely based upon special work as published in Mott's Archives of Neurology, Volume 2, 1903, carried out in the Claybury Pathological Laboratory during two years' tenure of the London County Council Research Scholarship in Insanity and Neuropathology. Mention has been made above of Tredgold's conception of primary and secondary aments. He regards the essential basis of primary amentia as cellular. Such gross conditions as porencephaly, hydrocephalus, microgyria, hemiatrophy, and the like, are of course frequently associated with amentia, but they are, according to Tredgold, accidental associations therewith. "The

essential basis of amentia is an imperfect or arrested development of the cerebral neurones." Tredgold calls attention to numerical deficiency in cells (compare Hammarberg's work), to irregular arrangement of cells and to imperfect development of cells, giving rise to the frequent appearance of certain cells first described by Bevan Lewis, or to some still more ill-developed cells which correspond with early neuroblasts. Tredgold states that the amount of change discoverable by the microscope is directly proportionate to the degree of mental deficiency present during life. Tredgold also ascribes importance to pigmentation, believing that it is an indication of defective metabolism in which anabolic processes cannot keep pace with the catabolic.

Hammarberg and Tredgold are accordingly in apparent discord as to the significance of irregular arrangement of nerve cells. Tredgold believes that his own experience as well as that of several others makes it clear that an irregular and haphazard arrangement of cortical cells is very characteristic of the condition. He states that such irregularities are found often where there is no accompanying sclerosis. This problem of the irregular arrangement of nerve cells and its significance accordingly remains a problem in the minds of many workers. Doubtless Tredgold would agree with Hammarberg as to the non-characteristic nature of the pigmentation found.

As to two points, Hammarberg and Tredgold are then in comparative agreement, namely, that numerical deficiency in nerve cells is a major feature in brains of feeble-mindedness and that the cellular appearances indicate imperfect development. If we consider the problem from the point of view of these two authors, accordingly, we must spend a maximum of attention on the number of cells and on the qualitative appearances therein.

We cannot pretend at this time to deal with the literature of the anatomy of feeble-mindedness with any completeness, although we have had the advantage of the relatively complete index of the literature of feeble-mindedness possessed by the Waverley School, from which we have abstracted over eight hundred references which deal pretty specifically with anatomical problems. We present below a selection from these anatomical references, having chosen those that relate more especially to the types of case considered in the first series of ten cases. The outstanding work of Bourneville and of Hammarberg as above mentioned, and the systematic treatment of Tredgold, do not by any means exhaust the anatomical interests of feeble-mindedness as presented in readily accessible literature. A book of strongly anatomical trend is that of W. W. Ireland called "Mental Affections of Children", wherein are given a number of points of view of permanent value. Ireland's classification has been given in brief above in the discussion of Tredgold's point of view.

Below we shall consider some American work in the field, before which it may be thought desirable to consider the status of the anatomy of feeble-mindedness as presented at the thirteenth international medical congress held in Paris in 1900. The eighth volume of the *Comptes Rendus* is devoted to psychiatry. There were three special questions proposed to the psychiatrists at this congress, one of which was the question of the pathological anatomy of idiocy. Reports by Mierzejewski, of St. Petersburg, of Shuttleworth and Fletcher-Beach of England, and of Bourneville, were presented, with a special contribution to histopathology by Philippe and Oberthür.

Mierzejewski considered that Bourneville's classification based upon coarse changes in the brain might well correspond to practical needs, but that a histological classification based upon special studies and upon exact embryological conceptions was sure to develop in the future. Mierzejewski considered that the basis of every anatomical lesion in the idiot's brain is a developmental deviation in the nerve tissue. Its origin must be sought in embryonic life or in disease occurring in earliest infancy. There could be no question of any true arrest of development without its morphological and histological basis in the entire brain. There could be determined, however, a true arrest of development in certain regions as indicated by the presence of neuroblasts. Mierzejewski went on to describe a particular group of cases, namely: those in which the white substance of the cerebral hemispheres is well developed but the gray matter of the cortex is distinguished by appearing in enormous and disproportionate amount. It was true that such brains belonging to microcephalic or demi-microcephalic subjects as a rule showed small convolutions (microgyria), but there were cases of this disproportionate development of the cortex in which there was no microgyria. Mierzejewski had published the first case of this nature in the *Comptes Rendus* of the international congress at Geneva in 1877.

The Mierzejewski type of idiot brain is characterized by a fairly developed centrum semiovale, an enormous distension of the ventricles, with the cerebral hemispheres transformed into a species of thin-walled vessel, 15 or 16 mm. thick. In such extreme examples as this, there is always microcephaly as well as microgyria, but there are other cases in which the white substance of the hemispheres is not so shallow and in which microgyria is absent. In such cases, finer methods will determine a lack or insufficient development of transverse intracortical medullated fibres. In von Monakow's case, there were neither any tangential fibres nor any striations of Vicq-d'Azyr. In other cases these structures were more or less well developed.

The situation was identical with the short and long subcortical association fibres, absent in certain examples, more or less developed in certain others. The radial bundles also varied in their development in these cases.

The gray matter presented at first sight the appearance of making up for the insufficiency of the white, sometimes presenting all its characteristic layers with perfect distinctness; in other cases presenting irregularity of arrangement.

The distinguishing feature of these hemispheres was the interpolation of a layer between the centrum semiovale and the cortex, of an intermediary strip of the so-called neuroblasts; or in certain cases, a layer of cells resembling those of the lower layers of the cortex, but irregularly arranged. In one case there had been interposed between the new layer and the overlying cortex a separate strip of white matter. This condition of affairs is called heterotopia of the gray matter. Although this particular form of heterotopia is diffuse, it does not resemble that more frequent form of heterotopia in which there are islets of ganglion cells scattered through the white matter. The microcephalic cases of microgyria show the interpolated layer to be formed of "neuroblasts." The demi-microcephalics have cells in the interpolated layer which resemble the so-called polymorphous cells of the undermost layer of the normal cortex.

This Mierzejewski type of microcephaly is accordingly distinguished from the ordinary microcephalic brain in that the proportions between gray and white matter, more or less well observed in the usual case, are not preserved in the Mierzejewski types. The demi-microcephalics according to Mierzejewski resemble the normal more closely in that the arrest of development on the part of the white matter is of slighter degree and is accompanied by a diffuse subcortical heterotopia of gray matter. But the principle is identical in both types of case.

Mierzejewski goes on to describe in more detail certain microscopic findings in a fresh case of the disease, and then sums up somewhat as follows: Idiocy may in certain instances accompany a condition in which the gray matter is extremely rich and in which there is an abundance of nerve cells. In these cases the commissural system of the convolutions is arrested in development so that there are not a sufficient number of paths of intercommunication and there is a disharmonious development of the different nervous elements. In the central nervous system, quantity alone does not count, but quality of elements and quality in their reciprocal combinations are the things that count. The white matter is nothing but a mass of prolongations of the nerve cells; accordingly richness of the dendritic branchings and of axis-cylinders of cells is the condition which most favors the plentiful development of white matter; and when these ramifications are few in number, the development of the white matter is insufficient. One of the chief conditions for irregular processes would then appear to be the multiplication and broad expanse of the connections of the pyramidal cells. Mierzejewski attributes to Dejerine the idea that intellectual supremacy seems to be the result, not so much of the number of cells,

as of the multiplicity and extent of their inter-connections. The idiot may have cells enough though these are deprived of their proper branchings. Mierzejewski throws out the suggestion that some of the neuroblasts lying below the normal-looking cortex may now and then proceed to a further development, and thus explain the occasional notable amelioration of mental faculties found in idiots.

The report of Shuttleworth and Fletcher Beach presents a classification of the pathological anatomy of idiocy under three main headings: (a) CONGENITAL; (b) DEVELOPMENTAL; (c) ACQUIRED or ACCIDENTAL. Among the congenital cases Shuttleworth and Beach enumerate eight forms.

The *microcephalic* form contains cases with cerebral hemispheres shortened in such wise as to reveal the cerebellum viewed from above, with such hindrance of development during intrauterine life (probably between the third and fourth months of pregnancy) as to disturb the later growth of the hemispheres as well as the detailed development of those parts which had been already formed. It is perhaps the temporo-sphenoidal and occipital regions which suffer most (as to the occipital region, Cunningham and Telford-Smith are quoted). Microcephaly is probably not caused by the premature synostosis of the cranial sutures. On the other hand, the development of the brain is probably the determining factor as to the form of the cranium (H. A. Humphrey). Microscopically one finds simply rounded or oval cells or cells with but few dendrites.

A second form of congenital disease is *hydrocephalus*, both that due to antenatal and that due to postnatal conditions. According to Meynert, congenital hydrocephalus extends the lateral ventricles lengthwise, while acquired hydrocephalus stretches the ventricles vertically and transversely. Cases of Bourneville, Beach, and Alexander Hill are quoted.

A third form of congenital disease is *scaphocephaly*, a condition possibly due to a premature union of the sagittal suture with an exaggeration in the growth of the coronal and lambdoid sutures in such wise that the head is elongated from before backwards, and may sometimes increase in height. But, on the other hand, this condition may possibly be the result of an original elongation and narrowness of the cranium, such as may be found in the natives of New Caledonia, the New Hebrides, and the Carolines. Sir W. Turner is quoted to the effect that this cranial form is due to intrauterine causes, such as inflammation or injury of the mother during pregnancy. Scaphocephaly does not necessarily produce idiocy, and an approximation of this form of skull may be found in North American tribes due to artificial compression.

A fourth type of congenital disease is the *Mongolian* type, with its special bony, cutaneous, mucosal, and cardiac conditions. Brachycephaly, approximately circular

in circumference, with a tendency to parallelism of the parietal and occipital surfaces, is noted. The skull is thin. There is an anomalously short little finger with lateral incurving displacement of the terminal phalanx. The skin is coarse and scaly, the mucous membrane irritable, the tongue is long, marked with deep folds, due to hypertrophy of papillae. Cardiac lesions are cited from cases of Garrod and of Thompson. Wilmarth's work is quoted as to the brains, which are said to show a very small pons and medulla, as well as very thin vessels. Shuttleworth thinks that these Mongolian idiots are essentially unfinished children, and that their singular appearance is that of a phase of foetal life.

The fifth form of congenital case is the so-called *genetous* or congenital neuropathic form, the offspring of neurotic parents. These cases are said to show very early in life excessive irritability, insomnia, attention disorder, and loss of self control. Such subjects are said to show microgyria at autopsy.

The sixth type is the type of *amaurotic family idiocy* of Tay and Sachs.

The seventh form is that of *sporadic Cretinism*.

The eighth form is a form presenting *partial and local atrophies*. Examples are absence of corpus callosum, porencephaly, cerebellar aplasia, and the like.

In addition to congenital cases Shuttleworth and Beach then consider the developmental group, under which they consider eclamptics, epileptics, syphilitics (including juvenile paresis) and paralytics.

They consider that the lesions found in *eclamptic* cases associated with the severe convulsions of dentition are local hemorrhages and adherent membranes which are inconsistent with the normal nutrition of the gyri. As for the *epileptic* group, the work of Bevan Lewis, of Andriezen, and of Echeverria is quoted.

Syphilis, they consider, is not a common cause of idiocy, but juvenile general paresis they regard as commonly caused by syphilis. They state that since Clouston first described this latter disease, there have been about 100 observations published.

The majority of the *paralytic* group of the developmental cases are of postnatal origin. These cases are attributed to inflammation of the brain or the meninges.

In the third group of accidental or acquired cases, Shuttleworth and Beach consider three forms: traumatic, postfebrile, and sclerotic.

The *traumatic* cases are due to compression of the head during prolonged labor or to the forceps. The lesions are situated as a rule in the Rolandic region. Small hemorrhages occur which are followed by degeneration and sclerosis. In the cases of cerebral diplegia, the most frequent cause is a meningeal hemorrhage especially affecting the veins between the pia mater and dura mater in the longitudinal sinuses, in such wise that the hemorrhage in finding its way into the great fissure compresses the upper borders

of the two hemispheres. The opinion of Osler and of Jacobi is quoted: that certain of these cases are actually due to foetal meningoencephalitis. Intelligence need not be markedly affected despite the severity of the physical disease.

Under the heading of the *postfebrile* cases, Shuttleworth and Beach also take up the cases of so-called hypertrophy. The most frequent acute infections which bring about these postfebrile cases are smallpox, typhoid fever, and scarlet fever. Meningeal, neuroglia, and nerve cell changes are noted in different degrees. The relatively rare affection known as hypertrophy of the brain shows an increase of neuroglia and of the white matter, but the nutrition of the brain seems also to be affected. The brain looks anaemic on section and strikes one as having been under a certain pressure within the skull, showing flattened and compressed gyri with sulci effaced. The most markedly injured part of the brain is the white matter of the hemispheres. The head does not reach the size of the head in hydrocephalus, nor is the increase so marked in the temples as it is in the superciliary region. The hydrocephalic head is rounded; the skull in hypertrophy tends more to squareness. In hydrocephalus the distance between the eyes is increased; not so in hypertrophy.

The third type is that of the sclerotic brains, carefully studied by Bourneville. Wilmarth is quoted as finding in 500 autopsies no less than 96 cases. The tuberous form of sclerosis was found in 15 instances.

Bourneville presented his anatomical point of view under the following ten heads:

1. Meningitic idiocy.
2. Meningoencephalitic.
3. Developmental (simple arrest).
4. Atrophic sclerosis.
5. Hypertrophic or tuberous sclerosis.
6. Porencephalic.
7. Pseudoporencephalic.
8. Myxedematous.
9. Congenitally malformed.
10. Microcephalic.

This account of the general aspects of brain anatomy in the feeble-minded might be concluded by a brief reference to the American literature. Besides the allied but separate topic of amaurotic family idiocy or Tay-Sachs disease, not here considered, and various case reports presented largely from the aspect of being interesting as clinical neurological phenomena by Mills, Spiller, Pearce, and others, the American literature is

devoid of much systematic work except that of Wilmarth about to be considered. From Massachusetts, a few cases have been published by Bullard and one by Southard. An interesting attempt was made by Orton in his "Pathological Study of a Case of Hydrocephalus" to parallel the cortex mapping of the topographers (Campbell, Brodmann, and the others) by a similarly conceived study of a markedly hydrocephalic case. He showed how certain portions of the cortex normally sunken below the surface are, as it were, ballooned out to appear on the surface of the dilated brain. The kind of work there initiated by Orton should be continued and performed in a large series of cases, but such work, of both an intensive and extensive nature, requires the resources of a brain institute to execute.

The largest series of cases of feeble-mindedness as yet studied in America from the anatomical point of view is unquestionably the series reported by A. W. Wilmarth, formerly of Elwyn, Pa. This work is favorably commented upon in many publications on feeble-mindedness.

Wilmarth reported in 1890 on the examination of 100 brains of feeble-minded children. The following table shows the classification employed although it is not now possible to learn which cases were entered twice in the table.

No actual disease or imperfect development of the brain	25
Non-development in various forms	16
Degenerative changes in vessels, ganglionic cells, or medullary substance not constituting true sclerosis	15
Sclerosis or atrophy	12
Diffuse sclerotic change	7
Tuberous sclerosis	6
No actual disease or involved development of brain but hypertrophy of the skull	6
Hydrocephalus	5
Extensive adhesion of membranes from old meningitis	3
General cerebral atrophy	2
Acute softening, recent	2
Demi microcephalus	2
Brain above usual weight but convolutions large and simple	2
Infantile hemorrhage	1
Angiomatous condition of cerebral vessels with degenerative changes	1
Glioma with sclerosis	1
Porencephaly with non-development	1

It will be noted from this list that no less than 31 cases, or at any rate 25 cases, were not observed to show any evidence of actual disease of the brain or of imperfect development therein. These observations are probably the most decisive in the literature upon which to found the idea that the brain of a feeble-minded person may not show in the

gross any evidence of defect. It is well known that in the field of mental disease proper, commonly known as insanity, the opinion long prevailed that there was not necessarily anything grossly abnormal to be found in the brains. Upon the older observations was founded the idea of the functional nature of mental disease so long prevalent. It would have been equally easy to have founded a hypothesis as to the functional nature of feeble-mindedness upon such data as these of Wilmarth. In fact, there are, so far as I know, no more modern data upon which to make a new judgment. In the somewhat allied field of epilepsy, however, Thom and the writer have recently discovered a somewhat similar percentage of normal-looking brains if we confine attention to the gross appearances; namely, about 30 per cent. Our own first series to be presented below (Waverley Research Series Cases I-X) shows at least three cases whose brains might well be considered in the gross as of a normal appearance. Perhaps, then, the generalization which Wilmarth's figures might permit will attain objective value. Wilmarth does not describe further these interesting cases with normal-looking brains. He states that 34 of the 100 cases belong to the school department or were employed as aids in the asylum department, and that 66 of the cases were children belonging properly to the asylum department.

Wilmarth was struck by the comparatively large number of cases of actual cerebral disease in contrast with the relatively small number with imperfect development which seems to be the causative agent. There were, however, 16 cases of so-called non-development in various forms, which put together with two cases of overweight brain with large and simple convolutions, a case of porencephaly with non-development, and possibly two cases of demi microcephalus, make a fairly large total of cases of arrest of development. As against these 21 cases of developmental arrest may be placed 42 cases; namely, 12 cases of sclerosis and atrophy, 7 of diffuse sclerosis, 15 of degenerative changes not regarded as constituting sclerosis, 2 of cerebral atrophy, 6 of tuberous sclerosis. It appears then that, although it is not safe to say that there is a relatively small number of cases with imperfect development, there is a somewhat larger number of cases of actual sclerosis of the brain substance which may be safely regarded as due in some sense to progressive changes, or at least to changes subsequent to birth. How many of these sclerotic cases are merely complications of developmental arrest was of course not possible for Wilmarth to decide and indeed constitutes a problem of the greatest difficulty. Besides the cases of arrest and the cases of sclerosis with or without arrest, there are a few cases of more obviously acquired nature, namely, one of infantile hemorrhage, three of old meningitis, and two of softening (though these latter may well have been cases of complication having nothing to do with feeble-mindedness).

The case of angiomatous condition of cerebral vessels was one of Mongolian idiocy.

Wilmarth had five cases of Mongolian idiocy in his series. He regarded the brains as "of good size for imbecile brains." The pons and medulla were very small, weighing about $\frac{1}{2}$ ounce in each instance, whereas the usual weight according to Wilmarth is nearly twice as much. He found the cerebral vessels inclined to be much thinner than in healthy brains, and was led to suspect from the defective nutrition and circulation of these children that the defective condition of the vessels was a general one. He laid down an interesting hypothesis in the following words: "From the small size of the pons and medulla in every instance, there seems to be a strong probability that the low nutrition, and possibly the other anatomical peculiarities of this group, may be due to the imperfect development or absence of certain cell groups in this region." The following extracts from Wilmarth's paper give further notions as to his conceptions:

"Probably the most frequent morbid condition met with is sclerosis in its various forms. The most destructive of these to the functional activity of the part involved is the so-called *sclérose tubéreuse*. It is usually seen on removing the membrane, as one or more areas of a white color and considerably harder in consistence than the surrounding tissue, slightly elevated above the level of the neighboring convolutions. It may be single but is usually multiple. It seems to be formed by a finely granular exudate, probably albuminous in character. If it occurs in the motor regions of the brain, the resulting paralysis is very complete, and if a large portion of the cortex is implicated, profound idiocy is liable to be the result."

"Another form of sclerosis, in direct contrast with that just described, is marked by shrunken tissue, the fibrous character of its structure, [*sic*] is more liable to be found in localized mass than in disseminated nodules, and is apparently less destructive to the function of the parts implicated unless it has reached an advanced stage."

"Non-development is found in several forms. A portion of the cortical substance may be thin, and instead of following the typical arrangement of the fully developed brain, form a number of irregular folds, which may be so small and numerous as to resemble a mass of angle worms. Again, the gyri may be of a normal size and appearance, but show a diminution in number, or absence of ganglionic cells in certain layers. Or, again, the convolutions may begin of normal size and development, but soon diminish and sink beneath the surface of the hemisphere, or perhaps entirely disappear, before acquiring near their normal length."

From the above all-too-hasty analysis of the literature of brain anatomy in the feeble-minded, we may suggest the following problems as facing us in the present situation.

1. There should be an attempt all over the world to swell *the number of properly reported cases* of feeble-mindedness *with autopsy*. If we speak of cases with suitable

histological studies alongside the clinical and anatomical observations, we shall shoot far over the mark if we concede that the literature contains 100 comparable cases — certainly a slender material from which to draw conclusions. Tredgold's interesting book, for example, is founded upon something like a dozen cases, among them epileptic and juvenile parietic cases. Hammarberg's work dealt with but nine cases. Many of Bourneville's cases are *curiosa*.

2. *Systematic external photography* and photography of the sectional brain should be freely used and reproduced. For in no other way can what is normal in the gross brain be established. The "normal" anatomists have little or no opportunity as a rule to lay down their own standards, using as they often do prison material, asylum material, and the like, and regarding "brain anomalies" with the same equanimity as anomalies in the liver.

3. *Total brain sections* may facilitate the just-mentioned task and are of especial value in marking off "secondary" from "primary" aments, through evidence afforded of special fiber-degenerations.

4. What may be called the "Mierzejewski effect" is worth particular attention, viz. the problem afforded by certain cases of disharmonious development of gray and white matter (gray in excess), in which the commissural system is thought to be deficient. Rich conscious endowment with poor mobilization might result in mild cases, if we carried the Mierzejewski idea to the limit.

5. From feeble-mindedness material we may hope to establish the truth or modified truth of the idea attributed by Mierzejewski to Dejerine, viz. that intellectual capacity depends more on cell interconnections than on cell numbers. The Dejerine argument is probably taken for granted by most workers nowadays but certainly needs proof and extension to details.

6. Hammarberg's idea that he could more or less approximately date the origin of many events of development in his cases requires further work. Such considerations as those of Donaldson *re* Laura Bridgman's brain are decisive here. Who knows what results bearing upon education would be obtained by exact ideas as to the chronology of normal cerebral development? The route to these observations may in part lie through feeble-mindedness material.

7. An exact definition in cerebral terms of "plastic impotence" (agenesia) and "plastic arrest" (aplasia) would help in many fields, especially perhaps in that of dementia praecox. Indeed strong arguments could be brought for the assertion that future psychiatry is to be founded upon a proper knowledge of feeble-mindedness.

8. The problem of the "normal-looking" brain in feeble-mindedness is intriguing.

Is there such a thing as "functional feeble-mindedness"? We speak of manic-depressive psychosis perhaps or of certain psychoneuroses as "discords played upon good instruments." Can the good instrument simply remain in its case, as it were, with resulting amentia?

9. A host of special problems in developmental mechanics, glandular dysfunction, disharmonious development of organs, heterotopia, dislocation of cells, reactive gliosis, satellitosis, premature pigmentation of cells, and the like remain, not to mention the problems of alcoholism and syphilis in parents and the operations of heredity working as it were *per se*.

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NOTE:—This bibliography is partial. It is based upon the entries in the card catalogue of the Massachusetts School for the Feeble-minded and contains those titles which specifically refer to the anatomy of the feeble-minded or articles which are known to contain anatomical material not indicated in their titles. To this fundamental list, which is indicated by Roman numerals and Arabic figures describing the place of articles in the Waverley collection, have been added further references called for by the present work. The bibliography is a working bibliography without pretensions to completeness, and has been arranged by years in such a manner that the history of the development of the subject is clearly shown. Future additions to this bibliography will be made, also by year numbers.

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